The 26th Annual Meeting of ADMA
Dordrecht 2016
Scientific Programme
Friday May 6 2016
Venue: Postillion Hotel

Welcome to the 26th Annual Meeting of the Anglo-Dutch Migraine Association!

Dear Colleagues,

On behalf of the Executive board of the Anglo-Dutch Migraine Association, as well as my fellow members of the Organising committee, it is my pleasure to welcome you to the Annual Meeting of the ADMA at Dordrecht.

We would like to thank the following for their support of this year's event:

Staff of Postillion Hotel
Jeannine Herzstein-Sijbring

I wish you a pleasant stay in Dordrecht and a joyful and fruitful scientific meeting.

Pjotr Carbaat, Local Organiser

**Morning Session**

From 8.30 Registration

9.00-9.10 Welcome and Introduction - Pjotr Carbaat and Emile Couturier

**Invited speakers**
Chairman: Emile Couturier

9.15-9.50 Spontaneous intracranial hypotension - Alok Tyagi, Consultant Neurologist, Southern General Hospital, Glasgow (UK)

9.50-10.25 Role of neuroimaging in spontaneous intracranial hypotension - Indran Davagnanam, Consultant Neuroradiologist, The National Hospital for Neurology and Neurosurgery, Queen Square, London (UK)

10.25-11.00 Coffee

Chairman: Sue Lipscombe

11.00-11.35 Sleep and headache - Hans Hamburger, Consultant Neurologist, Neurologie Centrum Amsterdam, MC Boerhaave, Amsterdam (NL)

11.35-12.10 CGRP, Sex and Gender in Translational Migraine Research – Antoinette Maassen van den Brink, Associate Professor of Pharmacology, Erasmus Medical Center, Rotterdam (NL)

12.10-13.30 Lunch

**Afternoon Session**

**Free Presentations**
Chairmen: Ton van Diepen and Manjit Matharu

13.30-13.45 Remarkable observations in a patient with the cluster-tic syndrome - Ilse de Coo (NL)
13.45-14.00 Head tremor and headache. Cervical dystonia? - Marit Hulzenga (NL)
14.00-14.15 Hull Prospective Analysis of OnabotulinumtoxinA (Botox®) in the treatment of chronic migraine; real-life data in 465 patients - Modar Khalil (UK)
14.15-14.30 Post Traumatic Headache: Misclassified, Mismanaged - Russell Lane (UK)
14.30-14.45 Electrotherapy and Headache: A Historical Perspective - Bart Lutters (NL)
14.45-15.00 Cumbria Headache Forum - Jitka Vanderpol (UK)
15.00-15.15 Pharmacological analysis of the increases in diastolic blood pressure and heart rate produced by (S) isometheptene and (R)-isometheptene in pithed rats - A. Labastida (NL)

15.15-15.40 Tea

**Closing Session**
Chairman: Emile Couturier

15.40-16.20 **24st Marcia Wilkinson Lecture**
**Novel tools and technologies to solve the mysteries of migraine**
*Zameel Cader*, Consultant Neurologist, John Radcliffe Hospital, Oxford (UK)

**Annual General Meeting**
16.20-17.30 Annual General Meeting (ADMA members only)

**Afternoon session - Abstracts**

## Remarkable observations in a patient with the cluster-tic syndrome.

*Ilse F de Coo[1], Joost Haan[1,2]*
[1] Department of Neurology, Leiden University Medical Center, Leiden, the Netherlands. [2] Department of Neurology, Alrijne Hospital, Leiderdorp, the Netherlands

### Background

The term 'cluster tic syndrome' is used for the rare ipsilateral co-occurrence of attacks of cluster headache and trigeminal neuralgia within the same patient (1). Medical treatment should combine treatment for cluster headache and trigeminal neuralgia, but is often unsatisfactory. In the literature, some patients are described who underwent surgical treatments procedures, which were often effective for both trigeminal neuralgia and cluster headache attacks (1-3).

### Aim

Here, we describe a patient who underwent microvascular decompression of the trigeminal nerve, primarily aimed at the 'trigeminal neuralgia' part of her pain syndrome.

### Case

In 2009, a now 42 year old woman started to suffer from pain paroxysms lasting several seconds to 2 minutes around the left eye and on the left forehead. After 6 months, she started to have redness of the eye and tearing during these attacks. These attacks, up to 30 a day, were never provoked by touching the face, talking or swallowing. One year later, next to the 'original' attacks, she developed a second kind of attacks in the same facial region, which were very severe, occurred up to 4 times a day, came in episodes of 2-3 weeks and lasted between 30 -90 minutes. During these attacks she had redness and tearing of

the eye, a ptosis and nasal congestion. A diagnosis of 'cluster tic syndrome' was made.
She was treated with a combination of verapamil, gabapentin, pregabalin and oxcarbazine. In 2015, she underwent a microvascular decompression of the left trigeminal nerve. Based on her last MRI, a compression of the trigeminal nerve by the petrosal vein was suspected. Indeed, during the operation, a compression by the petrosal vein was found, which was coagulated. Afterwards, both the frequency of trigeminal neuralgia and cluster headache attacks decreased. Moreover, the severity of the remaining cluster headache attacks decreased remarkably. Besides, she reported a shift from the left eye and forehead to the left temple.

**Discussion**

Although it is always difficult to draw conclusions from the description of one patient, the case described contains several remarkable aspects. First, a patient with a cluster-tic syndrome starting with trigeminal neuralgia without autonomic features, then developing trigeminal neuralgia with autonomic features, to end with the 'full' cluster-tic syndrome has not been described previously.
Second, the MRI did not show an arterial neurovascular conflict, but venous compression, confirmed during the operation. Venous compression as a cause of trigeminal neuralgia has been controversial, but recently reported in 38% of 326 consecutive patients who underwent microvascular decompression for trigeminal neuralgia (4).
Third, the response of the headache phenotype to the vascular decompression was remarkable. Efficacy of surgical therapy for the cluster-tic syndrome has been rarely described before. Invasive or destructive treatment aimed at the trigeminal nerve, however, has not been established in the treatment of cluster headache (5).

**Reference List**
(1) Alberca R, Ochoa JJ. Cluster tic syndrome. Neurology 1994;44(6):996-9.
(2) Solomon S, Apfelbaum RI, Guglielmo KM. The cluster-tic syndrome and its surgical therapy. Cephalalgia 1985 Jun;5(2):83-9.
(3) Watson P, Evans R. Cluster-tic syndrome. Headache 1985 May;25(3):123-6.
(4) Dumot C, Sindou M. Trigeminal neuralgia due to neurovascular conflicts from venous origin: an anatomical-surgical study (consecutive series of 124 operated cases). Acta Neurochir (Wien ) 2015 Mar;157(3):455-66.
(5) Miller S, Matharu M. Trigeminal autonomic cephalalgias: beyond the conventional treatments. Curr Pain Headache Rep 2014;18(8):438.

## Head tremor and headache. Cervical dystonia?

*Marit Hulzenga, Debbie Beumer, Peter J Koehler*
Neurology department, Zuyderland Medical Centre, Heerlen, The Netherlands

### Introduction

Head tremor is often encountered in the presence of either cervical dystonia (CD) or essential tremor (ET), and less frequently in other disorders. Various types of head tremor are recognized, among which a 'yes-yes' tremor that, in the presence of a more or less symmetric arm tremor, is highly suggestive of an ET. However, isolated head tremor is often misdiagnosed as ET. Recent literature suggests that the actual incidence of the combination is fairly low and that isolated head tremor is not part of the clinical entity of ET at all. Head tremor is commonly reported in cases with CD. CD with or without dystonic tremor, can be subtle and patients may be unaware of it.

In a recent review on headache and tremor we found that 'Although a dystonic ''no-no'' head tremor is frequently observed in cervical dystonia and headache in craniocervical dystonia is listed as a separate headache type in the International Classification of Headache Disorders (ICHD), we did not find any articles addressing their co-occurrence' (Kuiper et al. 2015). We aimed to describe a group of thirteen patients with a dystonic head tremor and their accompanying symptoms, to find out whether the presence of headache may be helpful in the differential diagnosis.

**Methods**

At our outpatient clinic we examined a 65-year old woman presenting with a headache from cervical muscle contraction that had been present for several years. Her headache was accompanied by a dystonic 'no-no' head tremor of which she was unaware. She was diagnosed with cervical dystonia and dystonic tremor with secondary cervicogenic headache. Based upon this case we analyzed data of 13 subsequent patients (9 women, 4 men; ± 64 years old) with dystonic head tremor (11 'no-no', 1 'yes- yes', 1 complex) presenting to our outpatient clinic to elucidate the issue.

**Results and conclusion**

Only four patients in our series had a combination of dystonic head tremor and cervicogenic headache of which three sought medical attention. The presence of headache in head tremor may point to dystonia; the absence of headache is not helpful in the differential diagnosis.

**Literature**

Kuiper M, Hendrikx S, Koehler PJ. Headache and Tremor: Co-occurrences and Possible Associations. Tremor Other Hyperkinet Mov (N Y). 2015 Jun 17;5:285

**Hull Prospective Analysis of OnabotulinumtoxinA (Botox) ® in the treatment of chronic migraine; real-life data in 465 patients**

*Modar Khalil, Hassan Zafar, Fayyaz Ahmed*
Hull and East Yorkshire Hospitals NHS Trust

**Introduction**

Chronic migraine (CM) affects 2% of the general population and is the most disabling form of the disorder with substantial impact on quality of life, oral preventive treatment for episodic migraine may work in CM, however poor tolerability and adverse effects are major limiting factors for its use, as well as a considerable number of patients do not respond.

The efficacy and safety of OnabotulinumtoxinA in adults with CM was confirmed in the phase III Research Evaluating Migraine Prophylaxis Therapy (PREEMPT) clinical programme leading to licensing authorities granting approval in CM. National Institute for Clinical Excellence (NICE) recommended OnabotulinumtoxinA use for CM in the National Health Service (NHS) in June 2012.

**Objectives**

To evaluate the efficacy and safety of OnabotulinumtoxinA in adults with CM in real-life settings.

**Method**

Adult patients with CM attending the Hull migraine clinic were offered OnabotulinumtoxinA based on clinical needs. All patients had tried and failed at least one oral preventive migraine therapy.

OnabotulinumtoxinA was delivered as per the PREEMPT study protocol and patients were asked to maintain a headache diary for at least 30 days prior to and continuously after OnabotulinumtoxinA (July 2010 and March 2015).

Data were extracted for headache days, migraine days, crystal clear (headache-free) days (primary outcome); also analgesic

medication overuse, Triptans use, and adverse events, before and after treatment (secondary outcome).
A responder was defined according to Hull Criteria (figure 1) as one with either
a 50% reduction in either headache days or migraine days or an increment in crystal clear days twice that of the baseline in a 30-day period (where pre-treatment crystal clear days were at least 3 days, otherwise patient has to achieve at least 6 crystal clear days post treatment to be a responder).
525 patients received 1880 cycles of OnabotulinumtoxinA, however we have included patients whom we have full data of their first cycle only (465 patients), as including subsequent cycles will skew the results of our analysis

**Results**

Full data were available on 465 patients (17.8% males), patients had the diagnosis of CM for a median of 4 years and 50.2% of patients were overusing various analgesics.
Statistical analyses showed significant improvement in all outcome measures tables 1, 2 and 3; table 4 summarises the commonest adverse events.

Table 1

| Outcome | N | Before treatment Median (IQR) | After treatment Median (IQR) | Change Median (95% CI) | P-value |
|---|---|---|---|---|---|
| Headache days | 465 | 27 (22, 30) | 18 (11, 27) | -6 (-7, -5) | <0.001 |
| Migraine days | 465 | 15 (10, 20) | 8 (4, 13) | -6 (-6, -5) | <0.001 |
| Crystal clear days | 465 | 3 (0, 8) | 12 (3, 19) | 6 (5, 7) | <0.001 |
| Mild days | 465 | 9 (5, 13) | 7 (3, 12) | -1 (-1, 0) | 0.001 |
| Painkiller days | 434 | 12 (7,20) | 7 (2, 12) | -4 (-4, -3) | <0.001 |
| Triptans days | 434 | 4 (0, 8) | 2 (0, 5) | 0 (0, 0) | <0.001 |
| Days off work | | 4 (3, 6) | 2 (0, 4) | -2 (-3, -1) | <0.001 |
| Outcome | N | Mean (SD) | Mean (SD) | Change Mean (95% CI) | P-value |
| HIT6 score | 349 | 68 (5) | 61 (8) | -8 (-10, -7) | <0.001 |

Table 2

| Outcome | ≥ 50% reduction N (%) | ≥ 75% reduction N (%) |
|---|---|---|
| Headache days | 126/465 (27.1%) | 53/465 (11.4%) |
| Migraine days | 216/465 (46.5%) | 72/465 (15.5%) |
| Painkiller days | 156/379 (41%) | 75/379 (19.8%) |
| Triptan days | 117/255 (45.9%) | 58/255 (22.7%) |
| Days off work | 36/59 (61%) | 23/59 (39%) |
| | ≥ 2-fold increase | ≥ 3-fold increase |
| Crystal clear | 211/465 (46.4%) | 119/465 (25.6%) |

Table 3

| Outcome | 50% responder | 75% responder |
|---|---|---|
| Any primary outcome measure | 272/465 (58.5%) | 150/465 (32.3%) |
| Any 2 primary outcome measures | 183/465 (39.4%) | 65/465 (14%) |
| All 3 primary outcome measures | 98/465 (21%) | 29/465 (6%) |

Table 4

| Adverse Events Observed | No of Patients N=465 (%) |
|---|---|
| Neck Stiffness | 74 (15.9%) |
| Pain at the site of injection for at least 24 hours | 68 (14.6%) |
| Ptosis | 42 (9%) |
| Reported but did not complain of inability to frown | 23 (4.9%) |
| Exacerbation of headache for five days | 18 (3.9%) |
| Difficulty in swallowing | 7 (1.5%) |
| Fainting during injection. | 3 (0.6%) |

**Discussion**

Our prospective analysis has shown that, in a real-life clinical setting, OnabotulinumtoxinA (Botox) can effectively reduce headache and migraine days, and increase crystal clear days from baseline.

Our cohort represent a more severely affected population than PREEMPT's as only a third of their patients had failed three preventive treatments; furthermore the pre-treatment headache days' number was higher in our patients.

**Conclusion**

OnabotulinumtoxinA is a valuable addition to current treatment options in patients with CM.

**Limitations**
A well-known effect in migraine studies is the high placebo response rate particularly with injectable treatment, the absence of an active comparator precludes comparison of the efficacy of Botulinum toxin type A with other therapies.

## Pharmacological analysis of the increases in diastolic blood pressure and heart rate produced by (S)-isometheptene and (R)-isometheptene* in pithed rats

*A. Labastida[1], E. Rubio-Beltrán[1], O. Hernández-Abreu[1], E. Rivera-Mancilla[1], B. Daugherty[2], S. Lederman[2], A. MaassenVanDenBrink[3], C.M. Villalón[1].*
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**Background**
Isometheptene (IMH) is a racemic drug with sympathomimetic actions that: (i) has been used for several decades in the acute treatment of migraine [1]; (ii) produces dose-dependent vasopressor and tachycardic effects in pithed rats [2]; and (iii) has been separated into the individual stereoisomers, (R)-IMH and (S)-IMH, with one believed to provide its therapeutic effect, while the other only contributes to side-effects (i.e. vasospasm) [3].
**Aims**
The present study has investigated in pithed rats the cardiovascular effects produced by intravenous (i.v.) administration of (R)-IMH and (S)-IMH as well as the pharmacological profile of the most potent stereoisomer.

**Methods**

Male Wistar pithed rats were prepared for recording of diastolic blood pressure and heart rate, as well as i.v. administration of compounds, as previously reported [2]. The effects produced by i.v. bolus injections (0.03-10 mg/kg) of (R)-IMH and (S)-IMH on diastolic blood pressure and heart rate were analyzed in control pithed rats. Moreover, the stereoisomer producing more pronounced vasopressor and tachycardic responses was further analyzed in pithed rats treated i.v. with the adrenergic antagonists prazosin ($\alpha_1$; 0.1 mg/kg), rauwolscine ($\alpha_2$; 0.3 mg/kg), the combination of prazosin plus rauwolscine or propranolol (β; 1 mg/kg), as well as with intraperitoneal administration of the monoamine depletor reserpine (5 mg/kg, -24 h).

**Results**

As compared to (R)-IMH, (S)-IMH produced more pronounced increases in diastolic blood pressure, whilst both compounds equipotently increased heart rate. The tachycardic responses to (S)-IMH were abolished after propranolol, but remained unaffected by the other compounds. In contrast, the vasopressor responses to (S)-IMH were abolished after prazosin or the combination of prazosin plus rauwolscine. Interestingly, in reserpinized rats, the tachycardic responses to (S)-IMH were abolished, whereas the corresponding vasopressor responses were partially attenuated and subsequently abolished by prazosin.

**Discussion/Conclusions**

The above findings, taken together, lead us to conclude that the tachycardic responses to (S)-IMH involve exclusively an indirect (tyramine-like action) mechanism, whilst its corresponding vasopressor responses are mediated by a mixed action involving an indirect (tyramine-like) mechanism and a direct stimulation of $\alpha_1$-adrenoceptors.

**References**
1. Diamond, S., & Medina, J., 1975. Headache, 15, 211-213.
2. Valdivia, L.F. et al., 2004. Life. Sci., 74, 3223-3234.
3. Lederman, S. et al., 2014. US Patent.
* (R)-isometheptene is being investigated in the US for tension-type headache under a US IND and is not approved for any indication.

**Post Traumatic Headache: Misclassified, Mismanaged**
*Russell Lane[1] and Paul Davies[2]*
Imperial College London[1] and University of Oxford UK[2]

Post traumatic headache (PTH) is recognised under IHC3β as a 'secondary' headache resulting from head and/or neck injury. We have examined risk factors for PTH and its management in a group of compensation claimants.
82 consecutive medicolegal reports written by one of us (RJML) were reviewed. The average interval from the index event to assessment was 21 months (range 5 - 77). 68 cases involved head and/or neck injury, most often resulting from road traffic (66%%) or workplace (11%) accidents. 6 claimants were non trauma cases and were excluded from further analysis.
Only 42 (51%) of the head and/or neck trauma claimants developed PTH despite the prospect of pecuniary advantage . 26 claimants (38%) with head and/or neck injury of similar severity to the PTH cases and 8 trauma cases without head or neck injury did not develop PTH. The head injury was 'minor' in 88% overall and in this small study, claimants with 'moderate' and 'severe' head injury were no less likely to develop PTH than 'minor' head injury cases. PTH was associated with neck injury alone in 7 cases (17%) but 7 non PTH cases also had isolated neck injury (26%). There was also no difference in neck injury severity between the two groups.

PTH conformed to IHC3β 'migraine' or 'probable migraine' in 62 (91%) of cases. 4 had migraine with aura attacks, and 2 had stabbing headaches and 2 trigeminal autonomic symptoms in addition to migraine symptoms. Only two cases had 'tension type' headache.

PTH had resolved in 17 cases at the time of assessment (9 within 6 months, 7 within 1 year, 1 in under 2 years) but 35 (83%) had ongoing episodic or chronic daily headache. 8 (19%) of these 35 cases had significant co-morbid psychological disorder compared to only 2 (7%) in the non PTH group. The frequency of pre-accident psychiatric disorder in the PTH (17%) and non PTH groups (15%) was similar but 83% of the PTH cases had a previous personal and /or family history of primary headache compared to only 4 (15%) in the non PTH group.

Less than half of the 42 PTH patients received any treatment for headache and only 15 were referred to secondary care. 19 received analgesics and /or anti-inflammatories but chiefly for neck and musculoskeletal pain but we did not attempt to identify cases of medication overuse headache (MOH) in this population. 15 PTH cases received migraine prophylactics, mainly amitryptiline but only 4 were prescribed triptans.

In conclusion, we believe PTH to be a *primary* headache disorder triggered exclusively by concussive injury to the head or neck and should be treated as such. The only clear risk factor for PTH is a previous personal or family history of primary headache. Reactive psychological factors and inappropriate treatment likely contribute to chronicity.

The prognosis for PTH at present is poor. This may be because many sufferers receive no effective treatment and are often dismissed as 'compensation seekers'. For the most part this attitude is prejudiced and unjustified.

**Electrotherapy and Headache: A Historical Perspective**

*Bart Lutters and Peter J Koehler*
Dept. of Neurology, Zuyderland Medical Centre, Heerlen, Netherlands

Chronic headache affects 3% of Western society.[1] Unfortunately, around half of these patients are resistant or intolerant to drug-therapy. Especially for patients suffering from drug-resistant migraine or cluster headache, neuromodulation may be a useful alternative. The medical use of electricity is, however, not as "high-tech" as is commonly presumed. In the first century AD, Roman physicians already made use of electric rays for the treatment of headache and gout, and with the rise of electrotherapy in the 18th century, various European physicians – such as Paets van Troostwijk and Elisabeth Garrett Anderson (England's first female physician) – started to use electrotherapy for the treatment headache and migraine.[2]

Not all scholars were optimistic about the electric cure. As early as the 18th century, the American polymath Benjamin Franklin argued that the positive effects of electricity in the treatment of paralysis were solely due to "suggestion". Later in the century, the Dutch pharmacist Willem van Barneveld (1717-1826) similarly attributed the positive effects of electrotherapy (at least partially) to "suggestion". Despite these critical notes, electrotherapy grew out to be immensely popular over the course of the 19th century. This changed at the close of the 19th century, when Leipzig neuropsychiatrist Paul Möbius (1853-1902) revived the view that the positive effects of electrotherapy resulted from nothing more than "suggestion".[3]

Möbius' claims were taken to heart by the Dutch physician Contant Delprat (1854-1934), at that time head of the electrotherapeutic ward at the *Binnen-gasthuis* in Amsterdam.

Delprat launched a research project, which compared the use of electrotherapy versus a sham procedure for the treatment of radial neuropathies. Electrotherapy did not prove more useful than sham-treatment and, subsequently, Delprat resigned from his post at the electrotherapeutic ward. Under the leadership of his predecessor, Johan Wertheim Salomonson (1864-1922), the ward slowly turned away from electrotherapeutics and by the 20th century had largely shifted towards the upcoming field of electrodiagnostics.[4,5]

During the first half of the 20th century, electrotherapy was largely abandoned by the medical profession. Möbius' statements turned out to be a prelude for the growing interest in the human subconscious, a view that was advocated by the famous Vienna ex-electrotherapist Sigmund Freud. In the age of psychoanalytic, the electric treatment of headache was abandoned, as its effectiveness continued to be disputed.[3]

Present-day use of deep brain stimulation (DBS), vagal nerve stimulation (VNS) and occipital nerve stimulation (ONS) significantly differs from early non-invasive applications of electrotherapy. Several upcoming neuromodulation techniques, however, such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS), largely resembles 19th century electrotherapeutics. Similar to earlier applications, these procedures do not appear to provide any permanent improvement and no valid evidence regarding temporal effects has been produced.[3] By putting these developments in historical context, we hope to demonstrate the need for placebo-controlled trials when analyzing novel forms of neuromodulation.

**References**

[1] Magis D, Schoenen J. Advances and challenges in neurostimulation for headaches. Lancet Neurology 2012;11:708-19.

[2] Koehler PJ, Boes CJ. A history of non-drug treatment in headache, particularly migraine. Brain 2010;133:2489-500.
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**Cumbria Headache Forum**
*Jitka Vanderpol MD FRCP BMA Cumbria Partnership NHS FT*

**Background**
Cumbria Headache Forum (CHF), established in 2013, provides regular large scale meetings regionally, open to all patients with headache and migraine as well as health professionals in Cumbria.
**Aims**
CHF enables access to medical professionals with an expertise in the headache field from Cumbria as well as invited experts from the outside of Cumbria. This is an educational platform aims to enable patients to take an active role in management of their often debilitating condition.
**Methods**
The concept combines pharmacological and non-pharmacological approach, including relaxation techniques, mind fullness exercises, lifestyle advice, stress management and diet. The sessions are run by headache experts, GPwSI, Headache specialist Nurses, psychologist, physiotherapist and dietary nurse, nutritional therapist, chaired by Dr Vanderpol Consultant Neurologist with expertise in headache field who heads Headache Service in Cumbria. To establish benefit and gather qualitative data a survey was conducted with

participants who attended headache forums between December 2014 and January 2016.

**Results**

In total 25 responded to the survey. 87% learned new information about headache or migraine, which has helped them to better understand the condition. 83% have taken more active role in management since attending the forum. 96% participants would recommend to family or friend who suffers from headache or migraine to attend the forum.

**Discussion and conclusion**

This concept provides multidisciplinary approach enabling and supporting Self-Management; the aim was to create a comprehensive program to increase the likelihood of successfully managing headaches and provide support to patients who often felt left alone for many years with their condition. Result of the recent survey has shown in overall very positive results.

Probably the best one day headache meeting in the world

Save the date for the 27th ADMA meeting in Norwich (UK):

**Friday May 26, 2017, arriving 25th.**
Local Organiser: **Alex Valori**

Website: http://www.anglodutchmigraine.org

Anglo Dutch migraine association

Founded in Maastricht, 1991